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TUBERCULOSIS IN ANIMALS OTHER THAN CATTLE

DISCUSSION BY THE CENTRAL VETERINARY SOCIETY*

Opening Contributions

I.—By R. E. GLOVER

In opening this discussion I would like to deal with some general aspects of the problem which would seem to emerge from investigations made during the past 50 years. I do not propose to cover the details of tuberculosis in non-ruminants and shall make little reference to the types of lesions produced in this disease since most members of the Society have had a far greater experience of the clinical manifestations than I. I intend to confine my survey to the animals of the town, *viz.*, the horse, the dog and the cat, and to pass under review four aspects of the disease ; incidence, evidence in favour of a decline in morbidity and mortality, diagnosis by tuberculin testing and responsibility to the public.

Incidence.—It is not necessary to give full details of the estimated incidence of tuberculosis since the general trends have been discussed at some length by other British workers (Dobson, 1930 ; Stableforth, 1929 ; Lovell and White, 1940). Tuberculosis in the horse is regarded as a comparatively rare disease. Between 1900 and 1918, the average figure based on *post-mortem* findings was 0·1 per cent. to 1·5 per cent. According to Calmette (1923) barely one animal in 15,000 passing through the abattoirs showed tuberculous lesions. Figures given by later workers are Verge and Senthille (1942a) 0·1 per cent., Oppermann (1941) 1·5 per cent. and Magnusson (1944) 0·9 per cent. It is of interest that Bywater (1949) recently has drawn attention to the rarity of the disease at the present moment, giving an incidence of three cases in 80,000 during the past six years in the district of West Ham. Thus it would appear that the disease is on the decline, at any rate in the towns.

Tuberculosis is more common in the dog. Between 1900 and 1904, 6 per cent. to 9 per cent. dying in veterinary schools were tuberculous. From 1900 to 1920 the incidence can be assessed at 4·2 per cent. Calmette (1923) and Lovell and White (1940) give figures of 0·4 per cent. to 1·0 per cent. for cases diagnosed clinically and 0·83 per cent. to 5·6 per cent. for autopsy material.

In cats Dobson (1930) reported an incidence of eleven cases out of 105 (2·2 per cent.) and Nieberle (1931) 18 in 241 (7·9 per cent.) ; later Lovell and White (1940) gave a figure of 3·6 per cent. Recent writers have indicated a level at the present time of about 2 per cent.

In considering these figures it must be borne in mind that the information on horses is based mainly on material taken at random from animals passing through slaughter-houses and may, therefore, represent a fair sample of the equine population. Nevertheless, abattoir figures must be accepted with some caution since, as pointed out by Vallée and Panisset (1920) the disease may arise in an " occult " form which is difficult to detect. It is possible that bacteriological examinations might reveal a higher incidence. The figures for dogs and cats, on the other hand, are largely derived from *post-mortem* material in veterinary colleges and similar institutes: they are thus highly selected and may fail to give an accurate assessment of the disease in carnivora. There is, nevertheless, a general impression that tuberculosis in cats and dogs is on the decline but more accurate figures would be of value.

With regard to the types of tubercle bacilli recovered from non-ruminants, it has been estimated that 80 per cent. of equine cases are caused by dysgonic strains of the bovine type, 10 per cent. to human and 10 per cent. to avian infections. M'Fadyean (1924) asserted that infection nearly always arises by the alimentary tract and that the disease usually is acquired in the suckling through the ingestion of tuberculous milk. In consequence, the primary lesions are located in the abdominal region. Occasionally rapid generalisation occurs with miliary pulmonary tubercles whilst the slowly progressive caseous lesions of the cervical vertebrae with resulting torticollis is a fairly common syndrome. In the cat, all but an insignificant proportion of cases are caused by the bovine type and the primary foci are abdominal—usually in the mesenteric glands. Thus Verge (1942b) summarising the literature reports 120 bovine strains from 121 cases. In the dog, on the other hand, about 70 per cent. are due to the human type. In a proportion the initial lesion is abdominal but primary respiratory foci are not rare. The incidence of the disease in dogs which frequent cafés has been remarked upon by French observers who believe that infection arises from infected sputum. The dog, however, is susceptible to airborne particles and it is not unlikely that aerogenous spread is responsible for the typical lung lesions.

There are three factors which must be taken into consideration in analysing any improvement in the situation: (*a*) a decline in the sources of infection, (*b*) the development of an enhanced resistance in the population as a result of long sustained exposure and (*c*) an alteration in the virulence of the organisms as a result of passage.

Sources of infection.—In cattle a deliberate attempt is being made to eliminate tuberculosis. Whilst there are no figures to show whether the campaign which is now being waged is beginning to produce results comparable to those achieved in the U.S.A., the steady increase in tubercle-free herds must have some effect in reducing the over-all incidence of the bovine disease. When we add to this the greater awareness of stockowners to the losses due to disease and their readiness to introduce hygienic precautions, and the widespread pasteurisation of milk, we

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can reasonably expect a steady decline in sources of infection by the bovine bacillus. We can anticipate, therefore, that the disease should become automatically extinct in cats: indeed any appreciable incidence of the disease in towns would indicate either that it is being transmitted from cat to cat or that human cases of bovine type are a menace to the cat. There should also be a reduction in equine cases, particularly in towns, but progress may be slower if foci of infection remain in pigs after the disease has been eliminated from cattle.

It is significant that Oppermann (1941) has found the disease to be more common in draught horses from country districts. A few cases of avian infection may still arise even if the bovine type is removed. It is perhaps of significance that I have been unable to find any records of equine tuberculosis in the U.S.A. during recent years and it is tempting to assume that the substantial progress made in the eradication of the bovine disease has eliminated the most common source of infection in horses.

In the dog, on the other hand, the reduction of bovine infection will not influence the incidence of tuberculosis to the same extent as in other animals. The predominant danger for the dog is the human being with an active pulmonary focus from which tubercle bacilli are disseminated in the sputum. We should expect, therefore, a gradual rise in the proportion of infections from human sources unless measures for the isolation of potentially dangerous human beings proceed as such a pace that this channel of infection is arrested as quickly as is the bovine source. For this reason it is important that, as far as possible, the type of bacillus responsible for canine cases should be typed.

Development of an Enhanced Resistance.—In the case of man, it is probable that resistance is a characteristic acquired through constant contact with the tubercle bacillus. We do not know how this capacity to withstand infection is "fixed" but recent evidence has suggested that an enhanced resistance can be transmitted hereditarily. The most significant data have come from studies of human twins who become tuberculous, carried out in the U.S.A. by Kallmann and Reisner (1943) and in Germany by Diehl and Verschlauer (1933). The former report showed that in 308 sets of twins which developed tuberculosis, the incidence in the monozygotic as compared with the dizygotic was as 1:3·5 and their corrected concordance rates 25·6 per cent. and 87·3 per cent. respectively. The hereditary transmission of a capacity to resist infection to tuberculosis has been placed on a firm footing by the experiments of Lurie (1941) who has been able to raise three pure line strains of rabbits with enhanced, moderate and low immunity respectively. These characteristics are fixed and are transmitted regularly to the offspring. The immunity is accurately assessed by giving graded doses of tubercle bacilli by the respiratory route. In the resistant group the disease usually progresses slowly. Anatomically, the syndrome resembles the type associated with reinfection in man and characterised by well capsulated cavities. Secondary foci produce a honeycombing of the lungs with cavities. In the susceptible group, on the other hand, the pulmonary lesions are more rapidly and uniformly progressive. Anatomically, the disease resembles a primary progressive infection with caseous foci showing little encapsulation. These findings have been confirmed by Diehl (1942).

In domestic animals no evidence has been produced to show whether there are variations in the susceptibility or resistance of races or individuals to moderate infection. In cattle it is known that tuberculosis is more widespread in certain African species than in others (Cornell, 1934), but there is no indication that "unsalted" cattle tend to develop a fulminating fatal disease, such as is encountered in man in the young adult native races which are exposed to infection through contact with Europeans. It would seem to be clear, however, that although the morbidity rate in European cattle, as evidenced by the tuberculin test, may be high, these animals must possess some degree of immunity inherited or acquired in early life, since most of them escape a severe progressive disease even when exposed to heavy infection.

In the horse, so few trials on the experimental induction of the disease have been undertaken that it is difficult to assess the resistance of the equidae. Eastwood (1911) put the matter to experimental test. He found that after the intravenous injection of 10 mg. of culture, the bacilli produced only slight tissue changes and did not form tubercles except in an occasional case where generalisation ensued. Vallée and Panisset (1920) held the view expressed earlier by Nocard that horses are highly resistant to exposure to experimental infection; they comment on the absence of any reports on transmission of infection from horse to horse. The apparent reduction in the incidence of the disease under natural conditions might be held to support the development of a state of acquired resistance, but there is no actual proof of any such development.

In the dog the observations of Cobbett (1907) are quite unequivocal and indicate a high degree of resistance to both human and bovine bacilli. For example, the subcutaneous injection or feeding of cultures was relatively ineffective since most of the animals showed little or no evidence of tuberculosis at *post-mortem* examination. The experiments of Gunn *et al.* (1943) are of particular interest. These authors infected dogs by the insufflation into the bronchi of small doses of tubercle bacilli. They draw attention to the regressive nature of the disease in some cases in which encapsulated caseous and calcified nodules were produced in the pulmonary parenchyma and regional lymph glands, while in others the disease was only slowly progressive and was characterised by necrosis and cavity formation accompanied by fibrosis. Reinfection with an equal or larger number of organisms 12 months later produced fresh pulmonary foci of the same nature as the primary lesion. The pathological findings closely resemble those described by Lurie in his resistant rabbits and are quite different from the rapidly progressive disease which is seen in guinea-pigs exposed to an airborne infection. It would appear, therefore, that in the dog the capacity to resist infection is not inconsiderable. It is very probable that this resistance is a sequel to its long and close contact with man.

Information about the behaviour of cats is still more meagre but in view of their almost complete insensitivity to the human type and comparative resistance to bovine organisms we must assume that they, too, are not readily infected.

Alterations in virulence as a result of passage through a host.—The horse is unique in that it is the only known mammal which apparently can induce a debasement in virulence of the tubercle bacillus with comparative ease. We owe most of our information on the character of strains isolated from the horse to M'Fadyean and Griffith. The latter (1937) summarising the results secured from 55 cases examined in England, reported that more than one half were caused by bacilli of low virulence. In his

own series of 24 cases he recovered 12 strains fully virulent for guinea-pigs and rabbits and 12 of sub-standard virulence, varying from very slight to considerable. The question arises, therefore, as to whether the horse acquires a strain of full virulence from cattle and that as a result of a long sojourn in the tissues of the new host, there is a gradual debasement in virulence ; or whether the equine disease is a separate entity in which strains of low virulence are transmitted from horse to horse. I venture to suggest that the former is the more likely explanation because (1) half the strains isolated are of the fully virulent bovine type and (2) no evidence has yet been submitted that tuberculous horses pass on infection (Vallée & Panisset, 1920). Griffith (1911) demonstrated by passage experiments in calves and rabbits that some aberrant types of equine tubercle bacilli could regain their full virulence.

It might be assumed that the attentuation in virulence of equine strains (which is also encountered in skin tuberculosis in man) is directly related to the high resistance of this species. There is reason to believe, however, that the ability to check infection is not responsible. For example, Cobbett *et al.* (1907) showed that after passage through dogs, which are comparatively resistant, the virulence of both human and bovine strains was unaffected.

The mouse and the rat possess a high degree of resistance to mammalian strains and require large doses of cultures to produce lesions which, if they are not fatal within a short time, undergo regression. The repeated passage of the murine-adapted strain through mice fails to effect any reduction in virulence for the guinea-pig. It is clear, therefore, that the aptitude to arrest the spread of infection in the tissues does not necessarily carry with it the ability to modify the virulence of the tubercle bacillus. It would appear, therefore, that in the horse some factor is operating which does not arise in other resistant species and it may be simply a question of time. In mice, passages have been made at fairly frequent intervals and the organism has never remained in the one host for long. In dogs and cats infection may be acquired in adult life so that little time is available for attenuation to occur in the body. In the horse, however, it is highly probable that in a proportion of cases the initial infection which is acquired when they are foals may lie dormant until much later in life: under these conditions, attenuation of virulence may arise. We can postulate, therefore, that those strains which show a debased virulence were originally bovine strains of full activity acquired early in life whereas those which on isolation are still of standard virulence were of more recent origin.

Tuberculin Test.—The concensus of opinion indicates that tuberculin tests in non-ruminants are not so accurate as in man or cattle. M'Fadyean, Griffith and Bang all used the subcutaneous test with some measure of success but there are many recorded cases of tuberculous animals which failed to react. The value of the subcutaneous test was carefully examined by the Royal Commission on Tuberculosis. Cobbett and Griffith (1913) found that whilst non-tuberculous horses were not responsive to a dose of 2·0 c.c. to 5·0 c.c., ten animals infected artificially all reacted. They regarded the test as quite reliable. There are few figures, however, on the general application of the test to horses. The same authors reported on 41 dogs infected artificially. Nine showed no lesions at *post-mortem* examination ; six were negative and three positive to tuberculin. Of 26 which were tuberculous, nine only were tuberculin positive. Tuberculin is, therefore, regarded as of little diagnostic value. In cats artificially infected, the results were better, giving five reactive out of nine tuberculous animals.

In the horse the intradermal test is claimed to be accurate if it is negative (Magnusson, 1944) but is erratic in tuberculous animals. Thus of 885 horses tested with bovine and avian tuberculin, 101 (11·4 per cent.) were positive to bovine tuberculin, 277 (31·3 per cent.) to avian, 84 (9·5 per cent.) to both. Plum (1945) as a result of 2,473 tests reached a similar conclusion. Oppermann (1941) who claimed an accuracy of 81·6 per cent., based his results on autopsy material in the Hanover Veterinary School as opposed to the general tests of the Scandinavian workers. There is, however, a striking lack of information founded on careful *post-mortem* findings as to the significance of the reaction to the intradermal test. The restricted *post-mortem* examination undertaken in an abattoir, especially if unsupported by a biological examination, may be misleading. It has yet to be determined how far in a resistant animal like the horse, insignificant lesions are overlooked and how far reactions to tuberculin may be due to sensitisation with other acid-fast organisms.

Considerable doubt has been cast on the significance of intradermal tests in the dog. The test has been used by practitioners and it would be interesting to know whether members of this Society have secured satisfactory results. It is evident that no real progress will be made until a series of cases can be subjected to tuberculin tests and then followed up by exhaustive *post-mortem* examinations. Under experimental conditions, however, a reasonable degree of accuracy seems to be attainable. Colwell & Mills (1940) infected dogs by the intratracheal route and then applied tuberculin tests at regular intervals using graded doses of O.T. ranging from 1·0 mg. to 0·001 mg., and PPD from 0·01 mg. to 0·00001 mg. It is clear from the protocols of these authors that, provided a fairly high amount of the active principle of tuberculin is used, good reactions can be obtained in a fair proportion of cases. Thus the strongest doses (1·0 mg. O.T. or 0·01 mg. PPD) gave unequivocal responses in 245 out of 341 tests with 25 doubtful when O.T. was used, and 241 positive with 22 doubtful in the case of PPD. They could find no evidence of a greater accuracy when PPD was used. It is interesting that the tuberculous dog appears to resemble cattle in requiring a relatively large amount of active principle to provoke an unmistakable response and differs from man and the guinea-pig, which are much more sensitive. I do not propose to enlarge on tuberculin tests since doubtless Mr. Paterson will be dealing with this aspect of the subject.

The Control of Tuberculosis from the aspect of Public Health.—Although the subject under discussion is tuberculosis in non-ruminants, one cannot avoid some reference to the disease in ruminants. There is a tendency to regard the control of tuberculosis in cattle as a purely agricultural problem. It is true that the gradual elimination of the disease from cattle is of vital importance to the stock-owner, but its suppression has repercussions in the sphere of public health. Myers (1940) has pointed out that " the disease can never be controlled in the human family unless it is controlled amongst animals with which man comes into contact and upon which he depends for a part of his food supply." The dangers of tuberculous meat and milk have been emphasised for the past 70 years and the many steps taken to effect a reduction in such sources of infection have undoubtedly played an important part in protecting man. The role of the non-ruminants in maintaining infection is less certain since it is not known how

far they are a potential source of danger to man. In all probability the horse can be disregarded on account of the relatively slight incidence of the disease, and the fact that the majority of cases are due to the bovine bacillus with lesions predominantly in the alimentary tract. The reported incidence of 10 per cent. human cases (Calmette, 1923; Verge & Senthille, 1942a) indicates a potential source of danger, but I know of only one recorded case of equine tuberculosis in this country which can be ascribed to the human bacillus (Griffith, 1936). Dogs and cats, however, which are in such intimate contact with man, may be responsible for the dissemination of infection. Nevertheless it is difficult to obtain concrete evidence of transmission of infection from these species to man and *vice versa*. The views of several authors have been well summarised by Lovell and White (1940) who point out that the danger cannot be ignored. The insusceptibility of the cat to human strains is reflected in the failure to secure clear-cut evidence of transmission from man to cats. Kuwabara (1939) followed up 30 cats kept in close contact with tuberculous patients for two to three years and found no evidence of the disease at *post-mortem* examination. In the dog the danger is probably greater. Stableforth (1929) reported that in four out of six tuberculous cases there was a history of human infection whilst Hjarre (1939) found six cases of human tuberculosis in 25 instances where the dogs in the families were tuberculous. Herlitz (1939) expressed the view that possibly one-third of all the cases of canine and feline tuberculosis originated from human sources, but that transmission in the reverse direction is less common than might be supposed. Nevertheless, so long as tuberculosis arises in household animals the intimate contact of these species with man must be regarded as a possible source of danger and the same degree of energy should be displayed in attempting to eliminate these cases and in drawing attention to the risk of the dissemination of infection by the non-ruminants.

References

Bywater, H. E. (1949.) *Vet. Rec.* **61.** 8.
Calmette, A. (1923.) *Tubercle Bacillus Infection and Tuberculosis in Man and Animals.* Williams & Wilkins: Baltimore.
Cobbett, L. (1907.) *Roy. Comm. Tuberc.* Pt. II. Appendix vol. II.
——, Griffith, A. S., & Griffith, F. (1907.) *Ibid.* Pt. II. Appendix vol. III.
——, ——. (1913.) *Ibid.* Final Rept. Appendix Pt. II.
Colwell, C. A., & Mills, M. A. (1940.) *Amer. Rev. Tuberc.* **42.** 271.
Cornell, R. L. (1934.) *J. comp. Path.* **47.** 320.
Diehl, K. (1942.) *Berl. Klin. Tuberk.* **97.** 331.
——, & Verschlauer, O. (1933.) *Zwillingstuberculose.* G. Fischer: Jena. (Quoted Kallmann and Reisner (1943).)
Dobson, N. (1930.) *J. comp. Path.* **43.** 310.
Eastwood, A. (1911.) *Roy. Comm. Tuberc.* Pt. II. Appendix vol. V.
Griffith, A. S. (1936.) *J. Hyg.* **36.** 156; (1937.) *J. comp. Path.* **50.** 159.
Griffith, F. (1911.) *Roy. Comm. Tuberc.* Final Rept. Part II. Appendix vol. IV.
Gunn, F. D., Mills, M. A., Shepard, C. C., & Barth, E. E. (1943.) *Amer. Rev. Tuberc.* **47.** 78.
Herlitz, C. W. (1939.) *Acta Tuberc. skand.* **13.** 125.
Hjarre, A. (1939.) *Ibid.* **13.** 103.
Kallmann, F. J., & Reisner, D. (1943.) *Amer. Rev. Tuberc.* **47.** 549.
Kuwabara, T. (1939.) *Kitasato Arch. exp. Med.* **16.** 275.
Lovell, R., & White, E. G. (1940.) *Brit. J. Tuberc.* **34.** 117.
Lurie, M. B. (1941.) *Amer. Rev. Tuberc.* **44.** Supplement.
M'Fadyean, J. (1924.) *J. comp. Path.* **37.** 44.
Magnusson, H. (1944.) *Acta. Path. Microbiol. Skand.* Supplement 54.
Myers, J. A. (1940.) *Man's Greatest Victory over Tuberculosis.* Charles C. Thomas: Baltimore.
Nieberle, K. (1931.) *Ergebn. allgem. Path. path. Anat.* **25.** 777.
Oppermann, M. (1941.) *Deutsch. tierärztl. Wschr.* **49.** 37.
Plum, N. (1945.) *Skand. VetTidskr.* **35.** 31.
Stableforth, A. W. (1929.) *J. comp. Path.* **42.** 91 and 163.
Vallee, H., & Panisset, L. (1920.) *Les Tuberculosis animales.* O. Doin et Fils: Paris.
Verge, J., & Senthille, F. (1942a.) *C.R. Soc. Biol.* **136.** 353; (1942b) *Ann. Inst. Pasteur.* **68.** 114.

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II.—By N. DOBSON,

Veterinary Laboratory, New Haw, Weybridge

In 1882 Robert Koch[5] announced his discovery of the tubercle bacillus. Many years before this important finding others had noted and given an admirable description of a disease in fowls, which leaves little doubt that the condition was tuberculosis.

Crisp[2], a doctor of medicine, reported in 1868 his observations, describing tubercles in the liver, spleen and on the peritoneal surface of the intestines of emaciated fowls. He attempted to transmit the disease by inoculating four pigeons with material prepared from the lesions of a fowl. Unfortunately he killed the pigeons six weeks later and found no lesions at *post-mortem* examination. In 1872[3], he reported that he had transmitted the disease into five hens by inoculation, all of which died 12 to 18 months later showing typical lesions at *post-mortem* examination.

In the same year Paulicki[11] made a report on 21 birds of various species from the Zoological gardens in Hamburg, and described a chronic disease associated with loss of weight and lesions in the liver, spleen, intestines, lungs and "lymph nodes" of the neck. He was unable to recover the causal agent but observed that the lesions in birds are the same as caseous pneumonia and miliary tubercles of man and apes and pearly disease in ruminants, with the exception that the condition in birds is more rapid and more widely disseminated through the different organs.

In 1875 Crisp[4] described the symptoms and lesions in 21 two years old pheasants but was unable to demonstrate the bacterial nature of the condition.

Sutton and Gibbes[14] recorded in 1879 their finding of tuberculosis in two birds and from 1881 working at the Zoological Gardens in London they reported many cases of the disease.

Following the discovery by Koch of the tubercle bacillus, Sutton and Gibbes[14] demonstrated the organism in tissues from affected birds. Before their findings were published Ribbert[12] of Bonn, Germany, reported that avian tuberculosis was caused by an organism similar to the tubercle bacillus which affected human beings. He failed to transmit the disease by feeding the droppings of severely affected hens, but by inoculating two fowls intraperitoneally with material from an intestinal lesion of a fowl, he succeeded in transmitting it. Naturally following the isolation of the organism reports from many countries record the presence of tuberculosis in various species of birds.

Koch[6], however, was of the opinion that all forms of the tubercle bacilli were identical and this belief he maintained until the International Congress on Tuberculosis in London in 1901; when he declared that he considered tuberculosis of poultry quite dissimilar to tuberculosis of human beings and unlike that of cattle.

Before Koch had reached this decision Rivolta[13] showed that the inoculation of the tubercle bacillus from birds into guinea-pigs caused local abscesses at the site of inoculation and failed to produce lesions in the internal organs. These findings were confirmed by Maffucci[8]. Straus and Gamaleia[15] carried out much original work on the character of the avian tubercle bacillus and Maffucci[8a] working at Pisa

reported his conclusions with supporting evidence that the organism causing tuberculosis in birds was quite a distinct variety from the one affecting man.

In 1907 Max Koch and Lydia Rabinowitsch[7] reported upon natural and experimental forms of avian tuberculosis in birds and mammals. They examined 459 birds in the Zoological Gardens in Berlin and found 25·7 per cent. affected with tuberculosis.

Following the British Congress on Tuberculosis a Royal Commission[16] was established which finally reported in 1911. The findings of this Royal Commission have nobly stood the test of time.

Van Es and Schalk[17] in 1914 published the records of their experiments with tuberculin as a diagnostic agent of tuberculosis in fowls. Many reports had been published of the value of tuberculin, using various techniques, but these workers employed the intradermal method recommended for cattle by Moussu and Mantoux[10] in 1908. This method has become accepted as the most efficient means of detecting tuberculosis in fowls.

The cause of tuberculosis in birds is usually the avian type of the tubercle bacillus. All species of birds appear to be susceptible to this infection and the infected bird is the greatest agent in the spread of the disease.

Calmette states, " On ne l'observe dans aucun pays du globe chez les espèces sauvages." This is of course equally true of all forms of tuberculosis.

The fowl, turkey, and farm-reared pheasant are all very susceptible, whilst the guinea fowl, pigeons, ducks, and geese when under free forms of husbandry are rarely found to be affected. In all these species of birds my experience would suggest that husbandry plays a great part in the spread of infection.

Tuberculosis in the members of the parrot family may be caused by the human, bovine or avian types of the bacillus.

To what extent is the avian type pathogenic for mammals ?—This type has been recovered from human beings, but the number of cases where the organism has been typed with certainty is exceedingly small.

Horses are susceptible to the avian type, and the findings of McFadyean[9] dealing with natural infection and others by inoculation will be well known to you.

In cattle and goats the organism rarely causes a progressive disease. These animals usually become infected when young ; lesions if present are often in the intestinal tract or associated lymph glands. When such lesions are present a positive reaction may result following the tuberculin test with avian tuberculin.

In swine there is a considerable incidence of tuberculosis due to the avian type ; the lesions frequently are associated with the lymph glands. This incidence has been quoted as high as 89 per cent. by Van Es and Martin[18].

Dogs and cats appear to be resistant to infection with the avian type, whereas rats and mice are susceptible.

Rabbits are readily infected by inoculation but not so guinea-pigs.

Symptoms of the disease in fowls.—The slow progress of the disease permits the bird to keep in good condition for a long period and few symptoms may be seen during the early stage of infection. Eventually the appetite fails, the bird becomes droopy, remains in or near the housing accommodation for shelter, a variable condition of diarrhoea may be present and the wattles and comb become pale and shrunken. A progressive wasting may occur. This is seen particularly in the wasting of the muscles attached to the sternum, but birds in fat condition at *post-mortem* examination, may have generalised lesions.

Lameness or stiffness, which may be of an intermittent nature, is often seen.

Lesions.—The liver, spleen and intestines are found regularly to be affected. When the disease is advanced it is usual to find the liver and spleen enlarged and showing multiple lesions varying in size from a mere whitish speck to some half an inch in diameter. In the liver these lesions rarely project from the surface of the organ, whereas in the spleen this commonly occurs.

Lesions in the intestines commence from the mucous surface, later penetrating the muscular wall and developing rounded, firm, white nodules projecting under the serous coat. The mesentery may show lesions.

In the lungs, lesions are not infrequent ; they may be small and found only when the lung tissue is cut, revealing firm masses which take on the form of a cast of the air vesicles.

The ovary, oviduct and testes are rarely found to be infected.

The bone marrow very frequently contains lesions ; the joints, joint capsules and tendon sheaths may be extensively affected. In advanced cases of tuberculosis the thymus and thyroid glands may be involved. Lesions in the heart are uncommon.

Method of infection.—The natural means of infection are by the digestive and respiratory tracts: in the former by consuming contaminated foodstuffs or water, and in the latter by the organisms being airborne on dust, etc. It is essential for the maintenance of good health that the housing accommodation should be well ventilated and crowding of birds strictly avoided.

It is improbable that the egg constitutes a serious factor in the transmission of the disease. The ovary, oviduct and testes are rarely found to be infected, and contamination of the egg shell with bacilli in the cloaca does not appear to be a dangerous source of infection, when one remembers the large number of healthy chicks which have been reared from flocks seriously affected with tuberculosis. In fact when groups of birds are found to produce a large percentage of positive reactors to the tuberculin test this is quite the best means of saving the strain of birds and producing a clean flock free from tuberculosis, provided the chicks are reared on clean ground in isolation.

There is a marked age incidence of the disease in an affected flock. In birds up to six months of age the tuberculin test may reveal no positive reactors, in those 18 months old 5 per cent., 30 months old 10 per cent., and in birds 42 months old and older the incidence may be 20 to 30 per cent.

The poultry industry, like other branches of agriculture, changes its customs and policy. In the early 1930's it was the custom to keep birds on breeding farms as long as they would live and produce a few good eggs for incubation ; later in the decade, pullet breeding became the custom, but now on many farms it is more usual to keep the birds about two seasons, with the exception of certain especially well bred stock on pedigree farms, which are kept many years.

Tuberculin testing.—In the fowl the best site for inoculation is the wattle, near the ventral border on the lateral surface. Using a fine dental type needle 0·02 c.c. avian tuberculin is injected as strictly intradermally as possible.

The formation of a small, rounded, firm nodule indicates a good intradermal inoculation. During the brief operation, the head of the bird should be held firmly in control, to prevent the necessity of more than one skin puncture, otherwise a trauma may occur which over the first 24 hours may cause a firm, rounded, dark-coloured nodule, which then tends to disappear. The tuberculin tested birds are examined at the 48th hour after inoculation. A negative reaction shows a perfectly normal wattle, whereas a positive reactor shows a diffuse swelling, which may be quite warm, and in a bird with a large wattle this may be more pendulous than its fellow wattle. In some birds the reaction may extend throughout the whole wattle, into the inter-wattle space and even into the uninoculated wattle.

When carrying out the test it is a good system to inoculate the right wattle of each bird, and when examining for the reaction to have the bird held facing one at a convenient level and using both hands, pass each wattle simultaneously between the thumb and first finger. By this means the detection of a positive reactor is made easily and efficiently.

The clinically affected birds which have become emaciated may not react to the test, therefore before commencing the inoculation the attendant should be warned to draw one's attention to all emaciated birds, and these should not be tested, but killed.

All positive reactors should be removed from the flock at once, and the non-reactors transferred to clean disinfected accommodation and clean land.

Good results have not yet been obtained by employing this tuberculin test on birds which do not possess wattles. All other sites and methods appear to have been tried.

Prevention of the introduction of the disease into a flock.—All adult stock should be purchased subject to passing the tuberculin test. Ensure that there is no overstocking in houses or on the land.

Keep houses and land clean and grass well cropped.

Water and food containers should be so constructed that the droppings and the feet of birds are excluded.

Never scatter food on the ground or in litter ; this is an excellent means of spreading diseases by contamination of foodstuffs.

Exclude all poultry from manure heaps. Keep pigs away from poultry, or land recently used for poultry.

Exterminate rats and mice and ensure these rodents cannot gain access to the food stores.

Carefully handle all stock regularly in order to gain information regarding their condition.

References

1 Calmette, A. (1922.) L'Infection Bacillaire et la Tuberculose chez l'Homme et chez les Animaux. 332. Paris: Masson et Cie.
2 Crisp, Edwards. (1868-69.) *Trans. path. soc. Lond.* **20.** 441-442.
3 ——. (1871-72.) *Ibid.* **23.** 312-315.
4 ——. (1874-75.) *Ibid.* **26.** 249-251.
5 Koch, Robert. (1882.) *Berl. klin. Wschr.* **19.** 221-230.
6 ——. (1902.) *Trans. Brit. Comm. Tuber. Lond.* **1.** 23-35.
7 Koch, Max, & Rabinowitsch, Lydia. (1907.) *Virchows Arch.* **190.** 246-541.
8 Maffucci, A. (1890.) *Zbl. alla. Path. path. Anat.* **1.** 409-416.
8a ——. (1891-92.) *Z. Hyg. InfectKr.* **11.** 445-486.
9 McFadyean, J. (1918.) *J. comp. Path.* **31.** 225-256.
10 Moussu, G., & Mantoux, C. (1908.) *C.R. Acad. Sci.* **147.** 502-504.
11 Paulicki, A. (1872.) Beiträge zur Pathologie der Vogel, Berlin. 67-68.
12 Ribbert. (1883.) *Deuts. med. Wschr.* **9.** 413-415.
13 Rivolta. (1889.) Quoted by Maffucci.
14 Sutton, J. B., & Gibbes, H. (1884.) *Trans. path. soc. Lond.* **35.** 477-481.
15 Straus, I., & Gamaleia, N. (1891.) *Arch. Méd. exp.* **3.** 457-484.
16 Tuberculosis, Royal Commission on. (1911.) London, Final Report, part 2, appendix 4. 168-191.
17 Van Es, L., & Schalk, A. F. (1914.) *Bull. North Dakota Agri. Exp. Stn.* No. 108.
18 ——, & Martin, N. M. (1925.) *Res. Bull. Univ. Nebraska Agric. Exp. Sta.* No. 30.

III.—By A. B. PATERSON*

Veterinary Laboratory, New Haw, Weybridge

Tuberculin is essentially the filtrate from steamed liquid cultures of tubercle bacilli, usually concentrated by heat and preserved by addition of chemical agents. The principle responsible for the inflammatory reaction when this filtrate is injected into the skin of the tuberculous subject has been suspected to be tuberculoprotein since the time of Koch, but unequivocal demonstration that tuberculoprotein alone was responsible awaited the work of Long and Seibert in 1926 and the years following. Their work culminated in the production of pure tuberculoprotein in dry powder form and the name Purified Protein Derivative (P.P.D.) was finally applied to this preparation.

At Weybridge, P.P.D. tuberculin is prepared from mammalian and avian strains of *M. tuberculosis* by their growth on synthetic medium and precipitation of the tuberculoprotein produced by means of trichloracetic acid. The protein is washed centrifugally by dilute trichloracetic acid and acid phosphate buffer, followed by re-solution of the protein in alkaline phosphate and addition of preservative. This concentrated solution, which is adjusted *p*H 7·0, is passed through a Sharples supercentrifuge to remove about 10 per cent. of antigenic protein of high molecular weight and the resulting concentrate stored in this form at 4° C.

When required for issue this concentrated solution is diluted with M/15 phosphate buffer containing 10 per cent. glycerol, 0·5 per cent. phenol and 0·5 per cent. sodium chloride so that its strength is 3·0 mg. protein/ml. in the case of " herd test " mammalian tuberculin and 0·8 mg./protein/ml. in the case of " herd test " avian tuberculin, passed through bacterial filter pads to ensure sterility and bottled in 1, 3 and 5 ml. phials. " Retest strength " tuberculin has protein contents of 1·5 and 0·4 mg/ml. respectively. Avian tuberculin is coloured red with an inert dye to avoid confusion with mammalian tuberculin while testing.

All tuberculin before issue has its protein content accurately checked by chemical analysis, is titrated in guinea-pigs against a standard P.P.D. tuberculin and tested in infected and tuberculosis-free cattle.

A P.P.D. tuberculin thus produced on synthetic medium has the advantages that:—

(1) All protein appearing in the medium during growth is of bacterial origin.

(2) Substances produced during bacillary growth and which might give misleading reactions are eliminated.

(3) Tuberculin can be accurately standardised by chemical means on its tuberculoprotein content, thus ensuring unvarying potency in successive issues.

Relatively little information is available on dosage of P.P.D. tuberculins in animals other than cattle. The doses given below must be regarded as being given for guidance and are tentative in nature.

* The method of production of Weybridge P.P.D. tuberculins was illustrated by means of a small scale demonstration.

Horse.—Retest strength mammalian and avian tuberculin can be given intradermally in the neck, the dose being 0·1 c.c. each. The increase in skin thickness should be read at 48 and 72 hours. If desired, a second injection of 0·1 ml.could be given at the 48th hour.

Pig.—Retest strength mammalian and avian tuberculin can be given in doses of 0·1 c.c. intradermally in the anterior border of the ear or base of the ear. In a positive case reaction may be evident, as inflammation at 24 hours, with discolouration, necrosis and ulceration at 48 hours or after. Mr. W. J. McIlroy, working at the Veterinary Laboratory, Weybridge, has obtained excellent reactions to both types of tuberculin in artificially infected pigs.

Dog and Cat.—Retest strength mammalian and avian tuberculin at dosage of 0·1 c.c. intradermally can be used. Results seem uncertain and if the animal is infected with tuberculosis the general systemic allergic reaction may cause death.

Rabbit.—Intradermal tests with tuberculin are not satisfactory.

General Discussion

Major Wilkins wanted to stress the matter of the intradermal test in horses. Dr. Innes and he had had an interesting case last year, details of which were about to be published, and as a result it was hoped to carry out a survey rather like those conducted in Denmark, Finland and Sweden. Claims were made in the text-books about the intradermal test in the horse which did not seem to be based on actual experience. Plum in Denmark in 1945 had come to the conclusion that the skin of the horse was very sensitive to tuberculin, due to previous infection with acid-fast bacteria, but the animal itself was resistant to actual infection. This was borne out by his limited experience. The tentative view was put forward that the intradermal test in horses was of little practical use.

Mr. Wright.—Thirty or 40 years ago they had many cats with enlarged glands due to tuberculous infection. He did a lot of cat practice and nowadays he found enlargement of the glands rare amongst cats; this might be due to the decline of tuberculosis in animals. In the dog he had seen a few cases but not recently. The last case he diagnosed was about 12 years ago in a wire-haired terrier—a case of pulmonary tuberculosis no doubt contracted from humans. He had met with four cases in the horse in the last 12 to 18 months; three were mammalian and reacted to the single intradermal comparative test and one was avian. The three mammalian cases were confirmed by *post-mortem* examination. One of the three was a Hackney pony which had been winning at various shows. It went lame in the shoulder and he noticed a lump like a tumour; this was blistered with potassium iodine internally but it remained. It was then decided to remove the tumour under a local anaesthetic. An incision was made and the lump dissected; it was a lymphatic gland greatly enlarged, and it was sent to Streatley; the examination report was of a definite lymph node due to mammalian bovine tubercle. That was removed in the November and the animal went through the winter, was shown and won in the spring. It then went off its food and wasted. After it reacted to the intradermal test he obtained permission to destroy it. It was remarkable that here was a show animal, with that large lymphatic gland, showing no signs of wasting, but that later it became just a waster and had to be destroyed. The *post-mortem* examination disclosed tuberculosis. The other cases he had mentioned were all confirmed on *post-mortem* examination. He had known horses which had reacted to avian tuberculin and were put on C.L.O. because they were wasting, which were now fat and round and fit to show and he was wondering whether to retest or let well alone.

Mr. Mitchell: He would like more information from Mr. Paterson on using only one tuberculin in testing pigs. He had information from the Ministry's laboratory and in dealing with herds of pigs in Essex he had had some where the reaction was avian and in others *vice versa* and it was rather risky.

Mr. Paterson.—The answer was straightforward. The advice given was based on the results of tuberculin tests on experimentally infected pigs. No experiments on a large scale based on field testing followed by *post-mortem* examination had yet been done by the Ministry.

Mr. Barton.—He had tested a number of pigs. He had asked the Ministry about avian and bovine and from the results he did not know which to believe. Since then he had tested with bovine alone but could not say whether the pig had passed the test or not.

Mr. Bywater.—He had no recent experience with the test in pigs but before P.P.D. or synthetic medium tuberculin he did a fair number of tests with the old types of avian and bovine tuberculins. His experience was that the reactions were larger from avian tuberculin even when the actual infection was bovine. That was before the refined products were available. Regarding tuberculosis in horses, he had received a paper from France on this subject and it would appear that no reliance was placed on the tuberculin test in horses. From the statistics quoted and the numbers of reactors in horses to the single intradermal avian and mammalian tests, it appeared that the number of reactors to avian was higher than to mammalian tuberculin. The animals were not examined *post-mortem* so it was not necessarily reliable. From his own experience of animals examined in abattoirs, few horses were affected macroscopically with tuberculosis. One could not get a true picture without figures from both slaughterhouses and knackers yards; figures from slaughterhouses were only one indication of the incidence. According to the French publication, in 1936 out of 67,507 horses examined in abattoirs only eight were affected; other figures were: 1937, 73,000 animals (12 affected); 1938, 63,000 (8); 1939, 48,000 (10); 1940, 36,000 (0); 1941, 11,000 (5); 1942, 4,000 (3); 1943, 5,000 (1); 1944, 3,000 (1); 1935, 11,500 (1); 1946, 31,000 (0); 1947, 60,000 (0). It would appear that the incidence of tuberculosis in horses was low and was declining. In 1922 he examined a large number of cows in slaughterhouses and over 75 per cent. had tuberculous lesions. Figures he had up to 1939 showed a marked drop in the incidence in cattle, due to the operation of the Tuberculosis Order and the fact that butchers were learning to be more careful in their purchases.

Mr. Dobson, reviewing tuberculosis in poultry, had said that it was largely a disease of older birds and that if poultry were killed off at two years the incidence was relatively low. In his experience tuberculosis in poultry occurred mainly on farms where poultry were not kept intensively but where they were more of a side line. Another source were backyards where they were confined to sheds and were none too clean, where the husbandry was haphazard and the birds were kept for a long period. He knew of two cases during the war where large houses were taken over and the aviary was used for poultry and they had tuberculosis, no doubt left by the birds which had been in the aviary before the war.

Professor McCunn said that he thought tuberculosis was common in horses; he had one in the institution at present. It did not turn its neck to the right as it should and they subjected it to a test. He had no faith in the intradermal test so he used subcutaneous and Mr. Dunn put the tuberculin (6 c.c.) in the animal. 'The horse's temperature was taken over a period of four days and the mean temperature was 98·9; the temperature at the ninth hour was 101·2; the 12th 101·6; the 15th 101·6; and at the 18th hour 101·2; next morning it was 98·9. Was that a reaction? If the horse had started off with a normal temperature and had shown that rise he would have said that he had got a positive reaction. On the advice of Mr. Glover he waited and four weeks after the first test the animal was tested intradermally with mammalian and avian; the mammalian gave no reaction at all but the avian gave about a 4 plus. They often found that a tuberculous horse turned like a boat in one piece. Another symptom in the horse was the passing of an abnormal amount of water. He had diagnosed tuberculosis in two horses because there was a peculiarity about their breathing. A good symptom was that on listening to the chest, they would find no marked respiratory adventitious sound but the animal would take a short, not a deep, breath and when it expired it did so as a horse with slightly broken wind. After expiration there was a little pause. This passing of much water (urine) and peculiar breathing were to his mind very suspicious symptoms. Mr. Bywater had raised the question of slaughterhouses and the incidence of tuberculosis as judged by the abattoir records. He agreed with him; he had done a lot of work inspecting horse meat going to Holland and France and he did not think he ever saw one horse carcase to be tuberculous. The point was, what was the incidence in these horses of any other disease? He instanced a case of a horse which had a liver of 100 lb. which was one mass of tuberculous corruption. The horse had been dispensed with not through illness, but because of its age; it was not 100 per cent. efficient. He knew of a chestnut horse which reacted subcutaneously and intradermally; it was treated with C.L.O. and was still going strong. He knew of other cases of horses which had got over the disability with C.L.O. He also knew a dog which when tested with a hypodermic syringe in the plural cavity, showed every smear crowded; he did his utmost to have the dog destroyed but the owner would not agree, so the dog's chest was drained and

it was placed in the country and given C.L.O. The dog made a reasonable recovery. He thought he would treat so long as the case was not an open one. He did not think they should delude themselves into thinking that all their wonderful measures had a 100 per cent. effect on the reduction of any sort of disease. If they made a chart they would find a high and low curve and that curve depended on the resistance and diminution of resistance. If there was a lot of disease the animals built up a great resistance and so in time the incidence became low. When the resistance was low the incidence went up. An example was provided by the American soldiers in this country during the war who were affected more rapidly and frequently than our men. He thought the Ministry was wrong in its methods of combating tuberculous; in tubercle free herds there would suddenly be a flare up though the animals might not have been in contact with a known case. If tubercle got into such herds there was catastrophe; what would happen in this country if the Ministry pursued its policy was that cattle would have no resistance to tubercle and it might bring chaos to the agricultural community.

Mr. W. McKay asked Professor McCunn if he meant that we should maintain tuberculous herds so that resistance could be built up; surely the object was the elimination of all tubercle. He was interested in the comment on C.L.O. and he did not doubt the diagnosis but he thought he needed to show that the findings were due to the dosing with the C.L.O. As to the incidence of tubercle in horses, in the north-east of Scotland, in about 25,000 cases sent to the knackery, he had only three cases of tubercle; one with a liver as described weighing about 1½ cwt. An animal that had not been touched on was the goat; he had seen two cases of tubercle in goats and he knew of a third. In some parts of north-east Scotland it was common for dairymen to keep goats for the prevention of contagious abortion; he knew of one case in a tubercle-free herd where there was no explanation for a breakdown; then the goat died and it was found that it was an advanced case of tubercle. As to tuberculosis in the pig, he had seen a fair amount in the slaughterhouse and the only time he had seen muscular tuberculosis was in that animal.

Mr. True.—He was not so sceptical of the C.L.O. treatment; he thought the vitamin played a great part. He had found that in the chronic condition of pyometra in dogs concentrated vitamin helped. On one farm he knew, a large number of sound animals were going down; they were bottle reared on cow's milk and the source of the lesions was obvious.

Mr. Pay: They had had a few cases recently from the Beaumont Hospital. One was a two-year-old mongrel which had an illness of eight weeks duration. Its history was peculiar. The illness started after a fall from a window. The dog showed pronounced dyspnoea with respirations 42. Mr. Knight examined the chest by X-ray and gave a tentative diagnosis of diaphragmatic hernia. A laparotomy was carried out and, as no hernia was present, the animal was destroyed. At *post-mortem* examination the condition of the dog was fair but on opening the thorax a peculiar appearance was observed; the right thorax was completely empty and the right lung collapsed and adherent to the roof of the thorax. The left lung was consolidated and a chronic granular pleurisy was present. A small caseous lesion was seen in the collapsed right lung. There was no apparent reason for the collapse as the main bronchi were normal. There were caseous nodules in the heart muscle. Smears were positive for acid-fast bacilli. In another case said to be suffeirng from fits, diarrohea and hard pad, the dog was destroyed as a suspected hard pad case. Examination showed infection with tubercle in the lung, liver, and mesenteric glands and yet the animal was only five months old. One other case of a Kerry Blue showed loss of condition for a year and it was X-raved by a medical practitioner who diagnosed a mediastinal tumour.. That dog was a case of tuberculosis with lesions in the glands, lungs and liver. He had seen several cases of tuberculous lesions in the cat; in several suspected cases of tuberculosis of the mesenteric gland the *post-mortem* examination showed an enlargement of that gland, but instead of it being caseous it showed a rubbery appearance with some tendency to lamination. Smears were negative. It was suggested that some of these cases were not of an infectious nature but were the result of an old haemorrhage in the gland with some degree of organisation.

Professor McCunn: Was it a fact that seagulls suffered rather badly from tubercle?

Mr. Dobson: There were many records in the literature that seagulls had been affected, but he had never found a seagull infected with tuberculosis.

Dr. Edwards: Listening to Mr. Glover, he could not but feel how deeply he had been impressed in his outlook by close contact with two great masters of the subject, namely Sir John M'Fadyean and Professor Vallée, having acquired the meticulous precision of the one and the inspirational gifts of the other. He (the speaker) was also much impressed by the mastery shown by Mr. Dobson of the very peculiar field of avian tuberculosis; as also by the clear exposition given them by Mr. Paterson of the fashioning of the weapon, namely, tuberculin, without reliance on which they had long come to realise there was no hope of successful attack upon the problem of tuberculosis as it confronted them in the domesticated animals.

He would like at that late stage in the discussion to make two general observations upon approach to the problem, the first of which was historical, and the second, comparative.

Dealing with the historical approach, he recalled speaking to the late Major Dunkin, shortly after his classic distemper researches had come to an end, about his proposed further field of research. To his surprise, Major Dunkin said he was contemplating taking up tuberculosis research. He (the speaker) vented his view that really that was an unpromising field to enter, as the Royal Commission on Tuberculosis had exhausted it. To which, Major Dunkin at once retorted: "That is the worst about it; people have been too apt to take it for granted that the Royal Commission closed tuberculosis research by completing enquiry, whereas in reality there is a great deal more that has to be found out." As events were to prove soon afterwards, Major Dunkin was right; he (Major Dunkin) himself was destined to contribute much to our further knowledge upon disease caused by a member of the acid-fast group, namely Johne's disease; they had now precise information upon the composition of tuberculin and the nature of the reaction provoked by it, instead of the entirely empirical knowledge they had previously; and unexpected findings, such as the widespread prevalence of tuberculosis among certain species of wild animals, such as field voles, all made them now cautious in expressing views regarding what future enquiry might still throw up if they ceased to look upon tuberculosis research as a closed book.

Secondly, dealing with the comparative approach, he had often been impressed by the narrowness of the knowledge displayed by medical men who were specialists in tuberculosis, their knowledge being confined to one species, and usually on the clinical aspect only of the disease as it came before them, without the necessary experience in *post-mortem* and laboratory investigation which led to a proper comprehension of the subject. Among veterinary surgeons, even though their attention was drawn at the moment chiefly to bovine tuberculosis, their outlook was always much broader because it was rare to find them laying down dogmatic opinions inasmuch as there fell within their experience the enormous variety of response to infection shown among the various species of domesticated animals. There was an enormous difference between human and bovine tuberculosis, which, when well appreciated, counselled entirely different methods for bringing the infection in the two species under control. But so there was between tuberculosis, for example, in the cow and in the horse, so that the veterinary surgeon was fully alive to the need for dealing with tuberculosis in a given species on its own particular merits: and, moreover, to the necessity for enlarging his outlook by comparative study of the disease in the different species.

To take, for example, the horse, he recalled how, in 1919, a cart-horse at the College having shown persistent debility, he (the speaker) decided to test it with tuberculin, by the I.D.P. test, and the animal gave a pronounced reaction. While on holiday soon afterwards, his chief, M'Fadyean, killed it, and finding no lesions, decided to have the bowels opened up and displayed in the manner then customarily employed by them in researches upon Johne's disease. The intestines showed, unexpectedly, lesions so closely resembling those of Johne's disease that they thought they had made a great new discovery. Laboratory tests, however, proved, in this classic case, that these lesions were caused by the avian type of tubercle bacillus. Again, recently, at the Cairo Veterinary School, an Arab filly was brought in with persistent extensive facial ulceration, and their mallein having failed to evoke a reaction, the animal was tested intradermally with mammalian and avian tuberculin, when it gave a typical reaction to the latter. To control the finding, a number of presumably normal horses were similarly tested, when it was found that nearly one half of them responded in a similar manner. Afterwards, *post-mortem*, the only lesions discoverable in the filly were some rare typical glanders nodules in the apices of the lungs, apparently caused by a strain of the *B. mallei* group not homologous with that used in preparation of the mallein, the skin ulceration being also accentuated by a highly necrotoxic and haemolytic strain of staphylococcus. What part, if any, an avian tubercle infection had played in the pathogenesis they were unable to determine as the avian bacilli were not recoverable by either cultural tests or small-

animal inoculation. Again, he recalled how in the days before and just after the first World War, at the College, in the course of their researches into Johne's disease, using only calves which had passed a test with avian tuberculin (which they had found to be as satisfactory as johnin in diagnosis and more easily prepared), those calves afterwards displayed extremely well marked reactions to the test at an interval after artificial infection with cultures of the Johne bacillus. However, on slaughter, to their surprise, they could find no trace of Johne lesions or of the bacilli at the usual seats of lesions whether by microscopic or cultural examination—yet, it must be that the bacilli had established themselves somewhere in the body.

In the human subject they had now come to realise that it was not the reactor who was most in danger, infection with the human type of tubercle bacillus being so widespread among the human population that by the time the subjects had reached maturity most had acquired an immunity (or "premunity") after an, usually "inapparent," infection. At some institutions, such as hospitals, where the staffs stood grave risk of infection from tuberculous patients, tuberculin tests were found necessary, to disclose, not the reactors, but the non-reactors, so that these could be immunised at once, with BCG, to evoke in them a "premunity" against virulent infection. This was just the contrary of what was the purpose in the testing of bovines, where it was the reactors that must be disclosed, and, of necessity, when eradication was the goal, eliminated.

Such were the kind of lessons to be learned from the comparative approach.

All went to show how extensive was the field still awaiting investigation. He could think of hardly any more profitable one for the ambitious young research worker to enter, with all the special discipline it called for in laboratory technique, animal observation, *post-mortem* and histological work, and immunity studies; and perhaps too, in the not remote future, new lines of chemotherapeutic enquiry which would alter profoundly our attitude towards established infection in at least some of the animal species.

The Replies

Mr. Glover.—The difficulty of interpreting the intradermal tuberculin test in the horse was the absence of a base-line. In cattle careful data have been acquired on the reactions of tuberculous and non-tuberculous animals to known doses of tuberculin protein: this information was not available for the horse. It might be that the horse was more sensitive than cattle, and in this respect was more akin to man. The doses employed in cattle might therefore be more liable to produce false reactions in horses. Practitioners who had access to studs of horses might try to ascertain what amount of tuberculin would produce negative reaction to mammalian and avian tuberculin: such information would be of great value in clarifying the significance of false positive reactions. With regard to the incidence of the disease, the figures obtained from abattoirs and knackeries would probably give a more accurate picture of the extent and distribution of equine tuberculosis than those secured in the colleges from selected animals. Nevertheless, the former might yet be misleading since tuberculous lesions in horses might be trivial and easily overlooked in the usual methods of meat inspection. Similarly the number of cases of canine and feline tuberculosis seen in the veterinary schools might be abnormally high and might not truly reflect the incidence in the general population. In regard to cats it would be of value to know if the incidence was less in the towns than in the country, as one anticipated that the pasteurisation of milk in the towns would result in a low incidence whereas in rural areas it might not show a significant fall for some time.

Mr. Paterson.—The experimental approach to the problem was very necessary and a real need. They did not know the effect of the different doses in pigs and the type of reaction in the different stages of infection. The work of Willis on guinea-pigs desensitised with tuberculo-protein and infected with tuberculosis showed that an entirely different disease picture might in infected desensitised pigs under different circumstances be produced. Death occurred at a much later stage than in non-sensitised pigs and there were no definite lesions found although many tissues were teeming with bacilli.

Mr. Dobson.—Whilst examining the carcases of cats received from the Public Health Department, Edinburgh, he found a number of them infected with tuberculosis. Many showed numerous lesions in the intestines; others had open wounds in association with the eyes and mouth. Tubercle bacilli were found to be very numerous in these lesions and all were of the bovine type.

CLINICAL COMMUNICATION

PREGNANCY FOLLOWING CAESAREAN SECTION IN THE COW

SIDNEY S. NYE, M.R.C.V.S.,

Romford, Essex

Subject.—Shorthorn × Jersey cow, second calver, due in ten days.

History.—July 25th, 1947 (10 p.m.). Cow in a field, laterally recumbent, slightly blown. She was treated with calcium borogluconate intravenously and left sitting comfortably.

July 26th, 1947. The cow being still unable to rise, a further calcium injection was given subcutaneously in the morning. By evening the cow was again laterally recumbent and showing symptoms of severe pain; the os uteri remained closed. Caesarean section was advised and carried out forthwith. Intravenous chloral anaesthesia was used (in the nine caesarean sections I have done under chloral hydrate anaesthesia I have not had cause for complaint); a flank incision was made, the muscles being broken down instead of cut (a method now discarded in both rumenotomies and caesarean sections as being unsatisfactory as regards healing). A live calf was removed from the right horn. Unfortunately, owing to fading light, there was not time to remove the ovary from the implicated horn, as I feel should be done, to minimise the risk of a further conception taking place on that side. Twenty minutes after completion of the operation, the cow rose and walked back to her box; recovery was uneventful, though the wound did not heal by first intention.

The os never opened; the cleansings were removed with the calf, but a large quantity of fluid remained in the uterus; this was absorbed and involution occurred normally. The calf was reared.

The cow was served in December, 1947, and during January and February, 1948. The uterus was irrigated with Lugol's iodine on March 16th and 25th, 1948, but subsequent (irregular) services failed to result in conception. In January, 1949, however, the cow got out and was served by a neighbour's bull. Pregnancy diagnosis in June and July confirmed pregnancy in the left horn. She calved normally on October 9th, 1949, with a live bull calf.

ABSTRACTS

[Procedures for Diagnosing Bovine Venereal Trichomoniasis and Handling Affected Herds. Bartlett, D. E. (1949.) *J. Amer. vet. med. Ass.* **94.** 293-305.]

Besides venereal transmission, infection can occur from artificial insemination from an infected bull, or through genital manipulation without due cleanliness. Infected bulls ordinarily show no symptoms, and remain permanently infected since no adequate treatment is available (Bartlett, D. E., "Experimental Treatment of *Tr. foetus* infection in bulls": Abstr. *Vet. Rec.* **61.** 151.). Details of diagnosis in the bull are given.

Samples of semen must be protected from extremes of temperature and drying, and examined within a few hours of collection, although they may still be satisfactory at 18 hours (preputial samples) or longer (vaginal samples).

Typically, vaginal infection lasts about four weeks and trichomonads are best detected 18 to 19 days after initial infection. Thereafter if cows return to regular oestrus early, trichonomads may be demonstrated two to three days prior to the next two or three heats. In animals with interrupted cycles, there is no such rhythm. Cows eliminate infection after the completion of a normal pregnancy, passing two oestrus periods, or resting 60 to 90 days *post-partum*.

Stress is laid on the treatment of trichomoniasis on a herd basis, since individual diagnosis of suspected females frequently is not possible. Alternative plans are suggested. Trichomoniasis should be suspected when most females require several services before recognisable pregnancy results (this may only be true of the heifers in a long-standing case), when the calving interval is markedly extended, when oestrus suddenly returns to females thought pregnant as a common occurrence, or when discharges and abortions occur.

D. H. L. R.

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[Semen Examination to Determine Fertility in Swine. Herrick, J. B. (1949.) *Vet. med.* **44.** 393.]

An estimated 10 per cent. of boars sold for breeding are later infertile, and the author, pointing out that the veterinarian can help breeders in cases of disputed fertility, gives a brief outline of his methods.

He uses an artificial vagina similar to that used on bulls, with a 150 to 500 c.c. flask to collect the large volume of semen, which ranges from 30 to 60 c.c. in young boars to 100 to 300 c.c. in older animals. The ejaculatory process lasts for up to 20 minutes, and consists of five parts, *viz.*, watery fluid and urine (5 to 20 c.c.), then milky fluid containing a high concentration of sperm, followed by clumps of gelatinous material resembling cooked tapioca and further portions of sperm-containing fluid and gelatinous material.

Several standard laboratory tests for semen are mentioned, but no instructions are given as to precautions to be taken during transport to the laboratory apart from straining through sterile cheesecloth to remove the gelatinous portion. Staining of sperm is mentioned but no specific stain is recommended.

Good quality semen shows active motility with mass movement and has less than 25 per cent. abnormal sperm. The density varies from 25 million to one billion per c.c.

D. H. L. R.

[This article is very sketchy, although sound enough as far as it goes. Much more detail is already available in this country, in the Commonwealth Bureau of Animal Genetics and Breeding publication, "The Semen of Animals and its use for Artificial Insemination," by James Anderson (1945).—*Abstractor.*]

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Passive Immunity in the Newborn Foal. Bruner, D. W., Edwards, P. R., & Doll, E. R. (1948.) *Cornell Vet.* **38.** 363.]

This article confirms previous reports that passive immunity is transmitted from the dam to the foal by the colostrum and not from maternal to foetal circulation through the placenta. Pregnant mares immunised with *Salmonella abortus-equi* antigen were studied as well as a series of mares that had produced foals with haemolytic icterus in previous seasons. The concentration of antibody in colostrum was shown to be considerably higher than the level in the mare's serum, but an active foal reduces the colostrum antibody level to about 5 per cent. of its original value within 12 hours. The milk retains this low concentration of antibody for several weeks.

The authors suggest that in foals deprived of colostrum at birth it might be desirable to give some level of passive immunity by means of blood tranfusions. It is also apparent that in the case of mares transmitting haemagglutinins, suckling of the colostrum by an older foal would reduce markedly the concentration of these antibodies so that the dam's foal could then be suckled ordinarily with less chance of disaster.

F. R. B.

REPORT

[Veterinary Department of Northern Rhodesia : Report for 1948.]

During the year under review there were, apart from contagious abortion and trypanosomiasis, 80 outbreaks of heartwater, 36 of rabies, 17 of blackquarter, two of East Coast fever, and two of foot-and-mouth-disease. Contagious bovine pleuro-pneumonia had not been seen since 1944. Lumpy skin disease (scheduled in April, 1948, under the Disease of Stock Ordinance) broke out in 24-head of Brown Swiss cattle immediately upon arrival from the Union of South Africa. Energetic measures successfully confined the disease to those cattle alone. Intravenous injection of formalin seemed to assist in suppressing secondary infection and sloughing. (Experiment on healthy station cattle showed that doses of up to 4 cc. of 50 per cent. commercial formalin per 100 lb. liveweight, suitably diluted for intravenous injection, could be given without other than transient ill effect).

Two further experiments using dimidium bromide (phenanthridinium 1553) were commenced during the year. " In the first, using the drug at the accepted Northern Rhodesia dosage level of 1·25 mgm. per kilo, *i.e.*, 1 c.c. of 3 per cent. solution per 50 lb. bodyweight, the purpose was to study the effect upon local cattle of repeated infection with *T. congolense* followed by repeated cure. Twenty cattle were used. The results of this work indicate that cattle can be subjected to such treatment without serious ill effect, and indeed, that they become more difficult to reinfect after four cycles of homologous trypanosome. In the second, an attempt was made to produce a strain of *T. congolense* resistant to dimidium bromide (P.1553) by means of treating cattle with subtherapeutic doses. At convenient dosage of the 3 per cent. solution, the dosage levels were 0·78 mgm.: 0·625 mgm.: 0·3 mgm. and 0·15 mgm. per kilo. During the experiment, again using 20 head, it was found expedient to stabilise the dosage towards the end of the work at 0·625 mgm. per kilo. The same Kalomo strain of *T. congolense* was found to have developed resistance to dimidium bromide since fresh susceptible cattle, inoculated eventually with pooled blood from the experimental stock, were not cured by the correctly administered standard Northern Rhodesia dosage of 1.25 mgm. per kilo, proved normally fully efficient, and actually almost twice the recommended minimal therapeutic dose of 0 87 mgm. per kilo. This work continues." Spray methods of tick control were described as attractive, the advantages including greatly reduced capital outlay in comparison with dipping tanks, mobility, and use of the dip solution at its optimal efficiency.

Sarcoptic, and to a lesser degree, demodectic, mange of cattle has almost disappeared from European farms where B.H.C. dips have been used over the last two years, though of unaltered incidence in at least one Reserve where there is no dipping. Streptothricosis (locally known as " senkobo skin disease ") has also lessened in similar circumstances.

During the year Government gave consideration to a growing tendency amongst European cattle owners to reduce their cattle holdings. The basic reason was understood to lie in the fact that beef cattle prices had not kept pace with the increase in prices of other agricultural products and this, connected with other difficulties in ranching, high mortality and shortage of wire, had led to a fall-off in interest and production. The trend was disclosed by the considerable disposal of breeding stock for slaughter, which found a readier market amongst butchers than amongst ranchers. In view of this, the Government approved a " Good Cattle Bounty Scheme," and at the same time drafted legislation to prohibit the slaughter of immature and breeding stock. The Scheme encourages livestock owners to carry out tick control and preventive inoculations.

Three consignments of hatching eggs, total 25 dozen, of the auto-sexing Legbar breed, were imported by air from England. Arrangements were successful in having one lot hatch out in time for the Agricultural Show in Lusaka where the day-old chicks attracted some attention. Poultry keepers in this country import very large numbers of day-old chicks from Southern Rhodesia each year, but as half the birds are cockerels and market conditions are not favourable to feeding cockerels there is a considerable wastage in freight and feed and general effort in raising birds to six weeks before the cockerels are eliminated. The Legbar breed is being examined with a view to avoiding this waste.

G. P. W.

ROYAL COLLEGE OF VETERINARY SURGEONS

Membership Examinations, December, 1949

List of Successful Candidates

(Concluded)

Glasgow

First Examination

Broadfoot, W. S.
Calder, Miss S. M. S.
Kerrin, F. E.
Mackintosh, T. M. S.
Watson, J. D.

Second Examination

Anderson, A. C.
Brown, P. R. M.
Cocozza, G.
Cunningham, M. P.
King, J. G.
Knight, T. A.
Kyle, David
Leach, P. A. G.
McCready, P. H.
McKean, Hugh
Taylor, A. M.
Taylor, J. W.
Todd, D. T.
Watt, W. R.

Third Examination

Anderson, A. W.
Bellinge, W. H.
Calder, R. H. (A)
De Boni, Miss R.
Dick, I. B.
Fitzsimmons, W. M. (H)
Hyatt, Miss M.
McIntyre, D. I.
McPhee, A. A.
Moffat, Robt.
Murdoch, M. L.
Randell, C.
Ricketts, Miss B.
Robertson, D. (A)
Stanhope, D. T.
Tattersall, D. J.
Waddelow, I. R. (H)
Walls, T. J. R.

(A) denotes credit in Anatomy.
(H) denotes credit in Hygiene.

Fourth Examination

Andrew, A. S.
Archibald, Miss H. I.
Clark, H. R.
Crighton, G. W.
Duncan, George
Ferguson, Thomas
Fraser, A. F.
Gallagher, Peter
Gross, Miss I. M.
Harris, G. D.
Heyworth, Brian
Hugh, A. R.
Macdonald, I. S.
Madden, Edward
McDougall, J. S.
McKerrell, A. L. L.
Ostler, D. C.
Patra, B. N.
Peritz, F. J.
Shepherd, Gordon
Sturdy, I. C.
West, W. R. G.
Wilcox, W. H.
Wilson, A. G.

Final Examination

Gilchrist, A. J. S.
Gillies, R. J. M.
Grisedale, J. A.
Grosset, A. B.
Horn, W. R.
Irvine, G. B.
MacInnes, D. I.
McAdoo, S. D.
Patterson, N. M.
Pugh, R. J.
Strang, A. L. D.

Dublin

First Examination

One subject

Cox, E.
Hernon, P. C.
Kelly, E. J. (C)
Magee, B. F.
McElroy, R.
O'Keefe, D. J.
Quirke, J. M.
Toolan, G. S.

(C) denotes credit in Chemistry.

Second Examination

Both subjects

Harte, J. E.
McGirr, P. J.
Smyth, P. W. W.

One subject

Agnew, J. H. (A)
Burke, J.
Caralon, E. S. E.
Casey, J. J. P. D.
Clarke, J.
Coghlan, D. P.
Conway, D. A.
Cosgrove, J. S.
Darcy, T.
Dougan, P. D.
Fitzmaurice, T. G.
Gallagher, E.
Gray, R. F.
Hart, Francis
Hatch, J.
Hurley, M. C.
Hurley, T. M. (P)
Keane, J. J.
Kirkpatrick, R. W.
Lee, D.
Lucey, D.
Lyons, T. M.
McDermott, Patrick
McGinley, M. D.
McLoone, A. G.
Mills, D. R.
Moscow, J. R.
Murphy, P. A.
O'Connor, Cormac
O'Loughlin, M. J.
Petris, M. A. (A)
Scott, W. A.
Waddell, A. R.
Wright, M. L.

(P) denotes credit in Physiology.
(A) denotes credit in Anatomy, etc.

Third Examination

Pathology only

Dunne, P. J.
Harney, J.
Lawther, S. McK.
Maher, M. P.
McAlinden, J. H.
O'Reilly, M. J.
Ronan, W.

Hygiene only

Casey, P. J. P.
Dowling, V. A.
Johnson, D. P.
Magee, P. J. H.
Nelson, W. A.
O'Connor, Michael
O'Connor, P. J. (Credit)
O'Driscoll, J. G.
O'Rourke, W. J.
Pyne, W.
Roche, L.
Roddy, R. M.
Schorman, N. S.
Scully, M. A.
Sheehan, D. P.
Taaffe, G. F.
Tyrrell, K. M. (Credit)

Fourth Examination (old syllabus)

Collins, Thomas
Keenan, N. T.
Leavy, J. J.
O'Callaghan, F. J.

Final Examination (old syllabus)

Barry, J. G.
Carroll, W. M.
Devlin, O. K.
O'Kelly, C. D.
Rooney, A. M.
Rosney, P. C.
Stewart, Paul
Thornton, I. M.

NOTES AND NEWS

Diary of Events

Jan. 5th.—Meeting of the Central Veterinary Society, at the Royal Veterinary College, Camden Town, 6 p.m.

Jan. 6th.—Meeting of the Southern Regional Group of the Association of Veterinary Teachers and Research Workers, at the Royal Veterinary College, Camden Town, N.W.1, 2 p.m.

Jan. 11th.—Meeting of the Editorial Executive Committee, 2.30 p.m.

Jan. 11th, 12th and 13th.—**Quarterly Meetings of Council, R.C.V.S.**

Jan. 13th.—R.V.C. Students' Society Annual Ball, Grosvenor House, Park Lane, W.1.

Jan. 18th.—Meeting of the Southern Counties Division, N.V.M.A., at Southampton (Polygon Hotel), 6 p.m.

Next Meetings of Council and Standing Committees, N.V.M.A.

(*Committees at 36, Gordon Square, W.C.*1)

Wednesday, February 8th

11.0 a.m. Parliamentary and Public Relations Committee.
2.0 p.m. Organising Committee.
4.15 p.m. Home Appointments Committee.

Thursday, February 9th

10.0 a.m. Veterinary State Medicine Committee.
2.0 p.m. General Purposes and Finance Committee.

Friday, February 10th

11.0 a.m. **Council Meeting** at the Connaught Rooms, Great Queen Street.

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PERSONAL

Birth.—Thomsett.—On December 7th, 1949, at The Gables Nursing Home, Aylesbury, to Babs (*née* Lay), wife of L. R. Thomsett, M.R.C.V.S., a son—Philip Lovell.

. . . . .

Forthcoming Marriage.—Vaughan—Lawson.—The engagement is announced between Leslie C. Vaughan, M.R.C.V.S., only son of Mr. and Mrs. E. C. Vaughan, of Sketty, Swansea, and Margaret Joyce, only daughter of Mr. and Mrs. J. Lawson, of Townhill, Swansea.

. . . . .

Death.—Cameron.—On December 17th, 1949, Mary Power, wife of John Cameron, M.R.C.V.S., 24, Lingfield Avenue, Kingston, Surrey, and daughter of the late William King and of Mrs. King, Cockermouth, Cumberland.

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R.C.V.S. OBITUARY

Lindsay, Peter Thomson, Capt. R.A.V.C. (S.R.) (retd.), 58, High Street, Woolton, Liverpool. Graduated Edinburgh, December 15th, 1908. Died December 21st, 1949; aged 62 years.

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LABORATORY ANIMALS BUREAU

Services Offered

As a supporting Society, the N.V.M.A. has pleasure in acquainting readers of its journal with the subjoined list of services offered by the Laboratory Animals Bureau which, it is thought, will be of interest to those whose work requires the use of animals.

1. *General Register of Suppliers.*—This is a list, complete so far as is possible, of suppliers who regularly, or from time to time, have laboratory animals for disposal. The information in it covers all species, and is available on request to all whose work involves the use of animals.

2. *Register of Special Varieties.*—This contains details of those who have notified the Bureau that they maintain stocks of special breeds, varieties, pure-line strains and the like. In not all cases are such animals freely available but usually a small breeding nucleus, at the least, will be offered to workers in particular need.

3. *Register of Accredited Breeders.*—A scheme is in course of preparation which will apply to guinea-pigs, rabbits and mice. Details will be notified later.

4. *Monkeys.*—A small number of monkeys of different species has been imported from West Africa during 1949. More are likely to be available in 1950, and enquiries are invited.

5. *Information and Advice.*—The Director is always glad to receive enquiries about the maintenance and breeding of laboratory animals. A considerable collection of reprints dealing with this subject exists at the Bureau.

6. *Catalogues of Invertebrates.*—Catalogues of arthropods, helminths, molluscs, and protozoa of interest in human and veterinary medicine were prepared and issued on request during 1949. These catalogues will be brought up to date and re-issued on request in 1950.

As from February 15th, 1950, the address of the Bureau will be: Laboratory Animals Bureau, M.R.C. Laboratories, Holly Hill, Hampstead, London, N.W.3. Enquiries should be addressed to the Director.

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Recent articles relating to the veterinary profession include "Veterinary Services in Great Britain" in the last issue of *The British Agricultural Bulletin* and "The Veterinary Profession in the United Kingdom" in the November issue of *The Kennel Gazette*. The former journal is published by the British Council quarterly at 3s.; the latter by the Kennel Club monthly at 2s. In the diagram illustrating the first-named article, "Ministry of Food" has been erroneously added in brackets under "Food Inspection."

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Sheep Scab:

Cumberland.—Linewath, Hesket Newmarket, Wigtown, Cumberland (Dec. 16th); Boot, Holmrook, Cumberland; Black Hall, Ulpha, Broughton-in-Furness, Lancs (Dec. 17th).

Lancashire.—Stock Farm, Colton, Greenodd, Ulverston, Lancs (Dec. 17th).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

THE PERSISTENCE OF PENICILLIN IN MILK

Sir,—While the technique of our experiments has not been the same as that of Zweig (1949), our results on a small series of tests lead to different conclusions. Comparison of the antibacterial activity of milk samples drawn at the regular milking intervals (twice daily) after injection of the quarters with 100,000 unit doses of (*a*) calcium penicillin in oil-wax; (*b*) procaine penicillin in oil-wax; (*c*) procaine penicillin in 2 per cent. aluminium stearate in oil, gave the following results in cows giving 20 to 30 lb. of milk daily.

Milkings after injection	Penicillin content of milk—units per ml.				
	(*a*)	(*a*)	(*b*)	(*b*)	(*c*)
1 a.m.	12·7	10·3	31·0	41·0	33·03
2 p.m.	6·07	5·2	13·7	10·3	5·4
3 a.m.	3·05	2·6	2·5	1·3	1·43
4 p.m.	0·86	1·3	—	1·3	0·65
5 a.m.	0·64	0·3	0·16	0·16	0·32
6 p.m.	0·08	0	0·21	0·02	0·01
Percentage recovery of penicillin injected	44·4	27·5	61·1	81	65·8

In our experience, therefore, the main result of injecting penicillin in the form of the insoluble salt is that a higher proportion of the dose injected is recovered, especially at those milkings immediately following the injection. With both the procaine preparations and the calcium penicillin, bacteriostatic concentrations persisted for five or six milkings, suggesting that injection once every three days might be adequate with any one of these preparations.

Some anomalous results were obtained with procaine penicillin in aluminium stearate, until it was found that some commercial preparations had widely divergent antibiotic contents.

Yours faithfully,

J. Carmichael.

Boarded Barns Farm,
Ongar, Essex.
December 16th, 1949.

Reference

Zweig, J. (1949.). *Vet. Rec.* **61**. 811.